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Application of press mud for soil amelioration

(press mud/water holding capacity/soil aggregation/pore space/rhizosphere)

D. PURUSHOTHAMAN

Department of Agricultural Microbiology, Tamil Nadu Agricultural University, Coimbatore-641 003

ABSTRACT Press mud an agricultural waste arising from the sugar mills has been studied under pot culture conditions for its suitability in improving the physical characteristics of red loamy soil. When applied at 5.0 t/ha, press mud increased the per cent aggregation, water holding capacity and per cent pore space. The effects on these soil characteristics were more pronounced under the influence of green gram. Therefore it is suggested as a soil ameliorant.

The fertility of soil is governed by a host of heterogenous factors like chemical, physical and biological properties. The physical properties of soil influence the chemical and biological properties very much. The role and importance of soil organic matter in improving the fertility of soils has been well understood[1]. In order to improve the organic matter content of soils, many agricultural wastes are being applied. Apart from adding to the organic matter level of the soils, these organic wastes improve the physical characteristics of soils.

Agricultural wastes like rice straw, wheat straw, coirpith waste, berseem guar and saw dust and several other wastes like paper mill waste, furnase dust, flyash etc. have been frequently used to improve the physical properties of problem soils. One of the solid wastes arising from the sugar mills, the press mud, has been recommended for soil application as an organic amendment. Recently, Karim *et al.*[2] emphasised the usefulness of press mud in supplying phosphorus to crops. In the present communication the influence of press mud application on certain physical properties of red loamy soil has been examined.

Press mud obtained from the Sakthi Sugar Mills, Coimbatore was dried, sieved through 100 mesh sieve. The red loamy soil of the Tamil Nadu Agricultural University campus was taken in 30 cm dia pots (5 kg/pot) and calculated quantities of press mud was incorporated into the soil to give 5 tonnes/ha level. The soil was maintained at 50 per cent of the moisture holding capacity throughout the experimental period.

Seeds of green gram (*Vigna radiata* Rox.) variety Co. 2 pretreated with *Rhizobium* sp. (cowpea miscellany group) were sown to the soil at the rate of 5 seeds per pot. Soil not sown to the crop but applied with the press mud was also maintained as control. The treatments were adequately replicated to permit statistical scrutiny of the data. At three stages of plant growth *viz.*, seeding, flowering and pod maturation stages soil samples were collected from each treatment. In case of soil with crop the rhizosphere soil samples were collected for examination[3].

Soil aggregation was studied using the Yoder's wet sieve apparatus with sieve openings of 8, 5, 2, 1, 0.5, 0.2 and 0.1 mm. Other physical properties like

TABLE 1

Effect of application of press mud on certain physical properties of soil

Treatments	Per cent aggregation			Per cent water holding capacity			Per cent pore space		
	1	2	3	1	2	3	1	2	3
Soil without crop (Control)	44.70	39.89	43.28	46.50	48.00	45.60	41.00	41.80	42.0
Soil with crop	45.80	46.59	47.32	46.62	51.65	56.72	42.72	43.80	45.46
Soil + Press mud without crop	40 23	45.34	46.11	67.00	67.90	68.22	50.16	50 40	50.55
Soil + Press mud with crop	45.69	45.79	51.11	54 63	58.53	64.00	46.15	48.75	50.06
	C D=2.86 (P=0.05)			C D=3.74 (P=0.05)			C D=2.24 (P=0.05)		

1. Seedling stage
2. Flowering stage
3. Pod maturation stage

water holding capacity, pore space and apparent density were determined using the Keen Raczkowski cup method[4]. The results are presented in Table 1.

It is seen from the results that due to the application of press mud, soil aggregation has increased considerably and it was well pronounced at the pod maturation stage of the crop. It is of interest to note that the rhizosphere soil has registered more per cent of aggregation than the uncropped soil. One of the explanations offered for the imporved aggregation in the rhizosphere region is that the microbial load and activity in the root zone might have been increased[5,6]. It has been well conceded that addition of organic amendments to soil increases the microbial activity[7]. Dakshinamurthi[8] pointed out that micro-organisms present in soils play a decisive role not only in the formation of aggregates but also in the stabilization of such aggregates. Humic and fulvic acids, microbial polysaccharides produced by micro-organisms were possibly responsible for aggregate formation and the water stability of the aggregates.

The water holding capacity of the soil added with press mud has increased strickingly. The data further revealed that for uncropped soil, the water holding capacity was greater than in the cropped soil. This may possibly be due to the growth of the crop, root penetration and also due to enhanced aggregation in the cropped soil.

The data on the per cent pore space revealed that press mud application increased pore space. It is interesting to note that in the cropped soil the per cent pore space increased with the age of the crop ; on the other hand in the uncropped soil the per cent pore space remained more or less constant. This clearly explains the root activity in influencing the pore space. It is perhaps that the root excretions of the growing plant improved the microbial activity and thereby brought about increased pore space.

Kanwar and Chawla[9] recommended the application of press mud to soil for the reclamation of saline and sodic soils. If we could utilize the abundant supply of agricultural and industrial wastes for any productive purpose of soil management, thousands upon thousands of hecteres of land lying barren for want of good physical properties can be brought under cultivation.

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Influence of *Aspergillus niger* on seed leachate content of bean

(seed leachate content/been/*Aspergillus niger*)

A. PRASAD* AND B. K. PRASAD

Postgraduate Department of Botany, Magadh University, Bodh-Gaya-894 234
**S. P. M. College, Udantpuri, Bihar Sharif, Nalanda*

ABSTRACT Electrical conductivity, soluble sugars and amino acids and germination of bean seed was observed storing the seed at 51.4, 63.3, 72.8, 80.0 and 90.7% relative humidity (rh.) at 30$\pm$1°C under the influence of *Aspergillus niger* for a period of 30 days. The electrical conductivity was found to be gradually increasing. Glucose, fructose and sucrose were detected in the leachate. Their amount was higher in the leachate of inoculated seed than uninoculated one adopting increasing tendency with increase in the rh. Amino acids L-Alanine, L-Serine, Phenylalanine, L-Leucine and unknown (Rf. 0.34, 0.42, And 0.47) that were detected, followed the similar trend of occurrence. One unknown amino acid (Rf. 0.42) was detected at 90.7% rh. only. The germination of seed remains least affected at 51.4 and 63.3% rh. while it obviously subdued at 72.8% rh. and above both normally in uninoculated seed and stimulated under the pathogenesis of *A. niger*.

Seedborne fungi deteriorate the seed in various ways. Permeability damage of the seed tissue is well established[1-4] with the consequence of leaching of soluble cell contents. Weber[5] has reported the value of electrical conductivity of the seed leachate proportional to the metabolite leached out of the seed, and accepted it as an index of extent of permeability damage of the cell. The leaching of metabolites adversely affects the seed health which is manifested as inverse correlation with germination[6]. Thus the value of conductivity may be conceiveed a parameter in determinig the deterioration of seed due to the activity of moulds. In the presen scheme, changes in the electrical conductivity of common bean (*Dolichos lablab* L.) seed leachate has been measured besides semi-quantitative assessment of the soluble sugars and amino acids induced by *Aspergillus niger* Van Tieghem and the seed germination.

A. niger was isolated from bean seed[7]. (ISTA 1966). 20 g of seed was inoculated aseptically with the fungus spores maintaining control of uninoculated lot after three months of harvesting the seed. The seed was stored over saturated solutions at 30 $\pm$ 1°C to maintain 51.4% ($MgNO_3$, $6H_2O$), 63.3% ($NaNO_2$), 72.8% ($NaNO_3$), 80.0% ($(NH_4)_2SO_4$) and 90.7% (KNO_3) relative humidities (rh) in sealed desiccator for a period of 30 days.

Measurement of electrical conductivity : 2 g each of the mentioned seed lots was surface sterilized with 2% NaOCl for 2 minutes and washed with distilled water and thrice with conductivity water. Seed so treated was taken to 20 ml conductivity water in broad tubes for 24 hours at 30$\pm$1°C for leaching of ions which was measured with conductivity bridge (Toshniwal make). Result of electrical conductivity was calculated substracting that of water[8]. (McKeen and McDonald 1967).

Determination of soluble sugars : 10 ml of leachate was concentrated at 60°C under reduced pressure to 1 ml. and sugar was analysed on thin

TABLE 1

Assessment of electrical conductivity, sugars and amino acids in the seed leachate and germination of seed under the influence of *A. niger* stored at varying relative humidities for 30 days.

% relative humidity	Condition of the seed	Electrical conductivity in 10^{-2} Mhos unit	Sugars: Glucose	Sugars: Fructose	Sugars: Sucrose	Amino acids: Alanine	Amino acids: L-Serine	Amino acids: Phenylalanine	Amino acids: L-Leucine	Amino acids: Unknown I Rf. 0.34	Amino acids: Unknown II Rf. 0.42	Amino acids: Unknown III Rf 0.47	% Germination of seed.
51.4	C	0.040	1+	1+	1+	1+	2+	1+	2+	2+	1+	-	98.5±1.67
	I	0.065	2+	1+	2+	2+	3+	1+	3+	3+	2+	-	94.7±0.95
63.3	C	0.045	1+	1+	1+	2+	2+	1+	2+	2+	1+	-	92.5±0.82
	I	0.070	2+	1+	2+	2+	3+	1+	3+	3+	2+	-	90.9±1.73
72.8	C	0.058	2+	1+	2+	2+	3+	1+	3+	3+	1+	-	85.2±2.01
	I	0.085	3+	2+	3+	3+	5+	2+	5+	5+	3+	-	65.6±0.95
80.0	C	0.075	2+	1+	2+	3+	4+	2+	4+	3+	2+	-	42.7±0.84
	I	0.103	3+	2+	4+	4+	6+	3+	5+	5+	4+	-	18.8±1.87
90.7	C	0.115	2+	2+	3+	4+	5+	2+	5+	4+	3+	1+	19.5±0.55
	I	0.145	4+	3+	5+	6+	7+	3+	6+	6+	5+	2+	0.0

C = Control
I = Inoculated
+ = Present, figure indicates semiquantitative grading
– = Absent.

layer chromatrogram[9] using Benzine : Acetic acid : Methanol (20 : 20 : 60) as solvent and Aniline-Diphenylamine-phosphoric acid as detecting reagent[10]. Sugars were identified with the help of standard and relatively quantified with the help of spot area and intensity and recorded using plus signs[11].

Determination of amino acids : Amino acids were determined[12] in the leachate concentrate used for sugar analysis in the same way using n-Butanol : Acetic acid : water (80 : 20 : 20) as solvent and ninhydrin-n-butanol-acetic acid as detecting reagent[13]. Identification and quantification were made as for sugars.

Germination of the seed : Seed stored at the noted rh was germinated in sterilized moist blotter taking 25 seeds in five replicates for each at 30±1°C and incubated for 7 days. Per cent germination was calculated.

The electrical conductivity of the seed leachate was found to be gradually increasing with the increase in the storing rh. The lowest value was recorded at 51.4% rh and highest at 90.7% rh.

Glucose, fructose and sucrose were detected among soluble sugars in the seed leachate. Their magnitude was higher in the leachate of inoculated seed than uninoculated one with increasing tendency with the increase in the relative humidity. Their minimum value was observed at 51.4% rh and maximum at 90.7% rh.

Soluble amino acids in the leachate were L-Alanine, L-Serine, Phenylalanine, L-Leucine and three unknown (Rf. 0.34, 0.42 and 0.47). These

were found to follow the similar trend of leak from the seed as for sugars. Unknown II amino acid (Rf. 0.42) was detected at 90.7% rh only. Phenylalanine also leached in relatively lesser amount.

The higher value of electrical conductivity of the leachate from the inoculated seed reflects the permeability damage of the cell with the consequence of uninterupted leaching of cell contents[1-4,14]. They have got direct correlation of electrical conductivity with ageing and diminished vigour of the seed that may be due to the consequence of environmental factor and microbial invasion[15], and remained inversely proportional to seed germination[6]. As permeability damage causes leakage[16] of K, Ca, Mg, P, amino acids and soluble sugars mineral deficiency and metabolic imbalance during germination is apprehended. Leaching of soluble carbohydrate and amino acids may lead to direct hunger of the seed[17] and the Cotyledons may not provide requisite nutrition to the seedling.

The higher value of the particulars of the scheme at increasing relative humidity even in the control seed indicates advanced deterioration of the seed at increasing relative humidity that becomes more stimulated and obvious due to *A. nigher*. Leaching of an amino acid (Rf. 0.42) at 90.7% rh indicates hydrolysis of protein into amino acid at this rh. normally in uninoculated seed and stimulated under pathogenesis. Deeper biochemical investigation is needed for such change in the seed at higher rh.

The seed germination is least affected at 51.4 and 63.3% rh even under the influence of *A. niger* which may be due to the equivalent moisture of the seed not favourable at these rh. Affects on germination is most adverse and well pronounced at 72.8% rh and above. This is further stimulated by *A. niger*. The figure of germination *vis-a-vic* electrical conductivity, soluble sugars and amino acids reflects that the degradation of seed is accompanied by permeability damage with the consequence of leaching of more and more metabolites and therefore their relative and quantitative evaluation may provide a clue in understanding the condition of seed health incited either by environment or the microbes at certain stage of storage as well as the condition of storage.

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Preparative and toxicological studies of some mixed ligand complexes of Hg(II)

(toxicological studies/mercury(II)–dien/amino acid/ligand complexes)

B. L. Bhat and K. P. Dubey

Department of Chemistry, University of Kashmir, Hazratbal, Srinagar-190 006

ABSTRACT Mixed ligand complexes of mercury(II), Hg (dien) $(ClO_4)_2$, Hg(dien) (en) $(ClO_4)_2$, Hg(dien) (lys)ClO_4, Hg(dien) (hist) ClO_4 Hg(dien) (cyst), where dien = diethylenetramine, en = ethylenediamine, lys = L-lysine, hist = L= histidine, cyst = L-cystine, were prepared and structures elucidated on the basis of elemental analysis and IR. The inhibition of acetylcholinesterase activity by the presently prepared and some other known compounds is also being reported.

The ligands (Pfaltz and Bauer, U.S.A.) were distilled before use. Other chemicals (B.D.H.) were of A.R. grade. The methods of preparation of different complexes are given below.

Preparation of Hg(dien)$(ClO_4)_2$: A solution of mercuric perchlorate was prepared by dissolving freshly prepared mercuric oxide (the required amount of mercuric oxide was precipitated quantitatively by adding excess of 2M NaOH solution to a solution of 9.05 g of $HgCl_2$ and washing the precipitate with distilled water by decantation) in minimum amount of warm 1 : 1 perchloric acid and solution filtered with a few washings.

Hg(dien)$(ClO_4)_2$ complex was prepared by mixing 40 ml of freshly prepared mercuric perchlorate solution with a solution of 3.433 g of dien in 25 ml of water. The reaction mixture was shaken well and then allowed to stand for two hours in ice. White crystals deposited were filtered, washed with cold water and ethanol and finally dried for 2–3 days at 40°C in an oven (yield=9.55 g).

Preparation of Hg(dien) (en) $(ClO_4)_2$, Hg(dien) (lys) ClO_4, Hg(dien) (hist) ClO_4, Hg(dien) (cyst) complexes : 5.026g of Hg (dien) $(ClO_4)_2$ were dissolved in 50 ml of hot water and mixed gradually with constant stirring with an equivalent amount of ethylenediamine (0.60g) or L-lysine hydrochloride (1.985g) or L-histidine hydrochloride (2.09g) dissolved in 10 ml of hot water ; or L-cystine (2.40g) dissolved in 40 ml of 1 : 1 perchloric acid respectively. In case of lysine system the resulting reaction mixture was evaporated to drynesswhen compound formed as faint yellow crystals. For other systems the white precipitate was directly obtained by mixing components together. The compounds so obtained were filtered washed with water, alcohol and ether and dried at 40°C in an oven. The yield was recorded to be 4.22, 3.92 and 2.50g respectively.

The other complexes[1,2] K_2 [(Hg$(TGA)_2$], Ca[Hg$(TGA)_2$], Na_2[Hg$(TSA)_2$] (where TGA = thioglycolic acid, TSA = thiosalicylic acid) were also prepared.

The mercury was estimated by thiosalt method[3] and sulphur was estimated by fusion and weighed gravimetrically as barium sulphate[4] (Table 1).

TABLE 1

Analytical percentage

S. No.	Complex	Solubility	% mercury T	% mercury F	% sulphur T	% sulphur F
1.	$Hg(dien)(ClO_4)_2$	Hot water	39.91	39.15	-	-
2.	$Hg(dien)(en)(ClO_4)_2$	Hot water	35.60	34.65	-	-
3.	$Hg(dien)(lys)ClO_4$	Hot water	36.50	46.00	-	-
4.	$Hg(dien)(hist)ClO_4$	Hot Na_2CO_3 solution	34.82	35.50	-	-
5.	Hg(dien)(cyst)	Hot NaOH solution	36.90	36.60	11.77	12.00

The IR spectra of the metal complexes in potassium bromide disc were recorded on Perkin Elmer Spectrophotometer in the region 4000 - 400 cm^{-1} (Table 2).

TABLE 2

IR absorption band frequencies of metal complexes (cm^{-1})

S No.	Complex	NH_2	Asym COO^-	Sym COO^-
1	$Hg(dien)(ClO_4)_2$	3310, 3250	-	-
2	$Hg(dien)(en)(ClO_4)_2$	3310, 3250, 2920	-	-
3.	$Hg(dien)(lys)ClO_4$	3420, 3280	1630	1400
4.	$Hg(dien)(hist) ClO_4$	3260	1650	1400
5.	Hg(dien)(cyst)	3440, 3260	1640	1400

A solution of mercury complex 0.003M was administered on 10% enzyme (rate brain homogenate) by homogenising tissue in 85% sodium chloride solution. The acetylchelinesterase activity was measured colorimetrically[5] and percentage inhibition calculated as :

$$\frac{\text{Experimental} - \text{Control}}{\text{Experimental}} \times 100$$

In case of en the NH peak is very broad lying between 3500 - 3200 cm^{-1} indicating extensive hydrogen bonding. In case of dien the peak appears as doublet at 3320 and 3260 cm^{-1} which are obviously due to presence of two types of -NH frequencies arising from $-NH_2$ (3320 cm^{-1}) and NH (3260 cm^{-1})[6]. The $-NH_2$ bond frequency for L-lysine hydrochloride and L-histidine hydrochloride lies between 3000 - 2800 cm^{-1} and this has been attributed to the existence of protonated[6] $-NH_2$. The amino acids also show bands corresponding to asym COO^- and sym COO^- stretching frequencies appearing in the range[7,8] 1630 - 1620 cm^{-1} and 1410-1400 cm^{-1} respectively.

TABLE 3

Inhibition of actylcholinesterase activity

S. No.	Complex	% Inhibition
1.	Hg(dien)(cyst)	85.60
2.	$Hg(dien)(lys)ClO_4$	62.60
3.	$Hg(dien)(en)(ClO_4)_2$	56.00
4.	$Hg(dien)(ClO_4)_2$	40.60
5.	$Na_2Hg(TSA)_2$	20.00
6.	$K_2Hg(TGA)_2$	18.00
7.	$CaHg(TGA)_2$	15.00

The IR spectra of Hg(dien) $(ClO_4)_2$ and Hg(dien) (en) $(ClO_4)_2$ complexes show $-NH_2$ bands shift to lower frequencies indicating coordination through nitrogen atoms of the ligand. In case of L-lysine, L-histidine mixed ligand complexes the NH_2 frequency is observed at 3420 cm^{-1} and in the range 3260 - 3260 cm^{-1}. The former may be due to free NH_2 group on lysine ligand and the latter which is an intermediate value[6] between NH_2 (3400 - 3500 cm^{-1}) and NH_3^+ (2800 - 3000 cm^{-1}) may be attributed to the coordination of amine to the metal ion. The shift in NH_2 frequency and the lowering of sym COO^{-1} stretching frequencies has been attributed to the coordination of mercury through nitrogen atom as well as carboxyl group of the amino acid. The IR spectra of mixed ligand complex of L-cystine shows band at 3420 cm^{-1} due to the presence of free NH_2 in cystine. However, the bands corresponding to sym COO^{-1} shift to lower

frequencies indicating coordination through oxygen atom of the carboxylic groups only.

A perusal of Table 3 indicates following order for the toxic effects of various types of mercury compounds.

Hg(II) mixed ligand complexes >Hg(II) simple complexes > Hg(II) complexes with sulphur donor ligands.

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Conductivity measurements and Walden product of some multicharged electrolytes in water

(conductivity/Walden product/multicharged electrolyte/water)

M. L. Parmar and Anita Khanna

Department of Chemistry, Himachal Pradesh University, Shimla-171 005

ABSTRACT Conductance measurements are reported for dilute solutions of aluminium amonium sulphate and potassium aluminium sulphate in water at 303, 308 and 313K. The Walden product $\triangle_m^o \eta_0$ has been calculated for both the multicharged electrolytes from the limiting molar conductance and viscosity of the water. The structure making/breaking ability of both the multicharged electrolytes has been inferred from the sign of $d \triangle_m^o \eta_0/dT$. Both the electrolytes have been found to behave as structure breakers in water.

Recently there has been a renewed interest in the conductance of aqueous solutions of unsymmetrical electrolytes[1,2], although the equations which are generally used for dealing with the conductance of symmetrical electrolytes are not totally applicable to unsymmetrical electrolytes.

Although number of conductance measurements have been reported in the recent past involving simple electrolytes in pure and mixed solvents, but literature provides very little information regarding the conductometric behaviour of multicharged electrolytes. Therefore, the present study has been carried out to learn more about the electrical conductivity of the highly charged electrolytes.

Aluminium ammonium sulphate and potassium aluminium sulphate were of A.R. grade and used as such without further purification, only after drying over P_2O_5. Fresh distilled water of specific conductance of the order of 10^{-6} Ω^{-1} cm^{-1} was used. All solutions were prepared by weight and conversion of molality to molarity was done by using the following expression[3]

$$C = d.\, m.\, 1000/1000 + mM_2 \qquad (1)$$

where C is the molar concentration of the electrolytes, d the density of the solution, m the molality and M_2 the molecular weight of the electrolyte taken to be as 453.33 and 474.44g mol^{-1} for $AlNH_4(SO_4)_2 \cdot 12H_2O$ and $KAl(SO_4)_2 \cdot 12H_2O$ respectively. The density was measured with an apparatus similar to the one reported by Ward and Millero[4], and described earlier[5]. Conductance measurements were carried out with the help of Naina digital conductivity meter Type NDC 732 at 50 Hz supplied by Naina Electronics (P) Ltd. The conductivity cell having cell constant 4.153 ± 0.001 cm^{-1} was used for the present study. All measurements were carried out in an air thermostat with a temperature control ± 0.01°C.

The molar conductance values of aluminium ammonium sulphate and potassium aluminium sulphate in water at 303, 308 and 313K are given in Table 1. It is evident from Table 1 that the molar conductance of $AlNH_4(SO_4)_2 \cdot 12H_2O$ as well as $KAl(SO_4)_2 \cdot 12H_2O$ decrease with the increase of concentration of the multicharged electrolyte. It is also

clear from Table 1 that the molar conductivity increases with the increase of temperature at a particular concentration.

TABLE 1

Molar conductivity of aluminium ammonium sulphate and potassium aluminium sulphate in water at different concentrations (C) and temperatures

C (mol l^{-1})	Δm (S cm^2 mol^{-1})	C (mol l^{-1})	Δm (S cm^2 mol^{-1})
Al $NH_4(SO_4).12H_2O$		$KAl(SO_4)_2.12H_2O$	
Temperature : 303K			
0.0099	242.70	0.0099	251.70
0.0197	227.24	0.0198	230.80
0.0394	204.25	0.0394	210.91
0.0589	188.84	0.0588	194.48
0.0779	175.97	0.0781	178.74
0.0973	164.34	0.0972	169.19
Temperature : 308K			
0.0099	272.67	0.0099	272.67
0.0197	249.97	0.0198	254.70
0.0394	221.35	0.0393	229.02
0.0587	205.17	0.0587	211.56
0.0779	186.59	0.0780	194.49
0.0970	173.40	0.0970	179.91
Temperature : 313K			
0.0099	293.65	0.0099	299.04
0.0197	273.07	0.0197	280.12
0.0393	237.77	0.0393	256.43
0.0596	222.98	0.0596	234.50
0.0798	200.56	0.0798	213.50
0.0967	195.41	0.0967	195.87

Because ionic mobilities, from transport experiments, are difficult to obtain for highly charged ions, which may form ion pairs[6], the first problem in treating the present data was to obtain a value for the limiting molar conductance ($\Delta_m{}^0$) for each electrolyte. At very dillute concentrations it would be expected, from Onsager's equation

$$\Delta m = \Delta_m{}^0 - S\, C^{\frac{1}{2}} \tag{2}$$

that a plot of the molar conductance Δ_m against the square root of the concentration C (molarity) should be linear and extrapolate to give the value of $\Delta_m{}^0$. However, Kay[7] has shown that for aqueous solutions of symmetrical electrolytes, $\Delta_m{}^0$, as determined by this method, will tend to be somewhat lower than the value obtained from the more complete Fuoss–Onsager equation[8,9]

$$\Delta m = \Delta_m{}^0 - S\, C^{\frac{1}{2}} + EC.\log_{10}C + J.\,C. \tag{3}$$

In these eqns. S and E are constants and are functions of fundamental physical properties such as dielectric constant, temperature, viscosity and ionic charge, while J contains these and also a distance parameter a, ostensibly the minimum distance of approach between ions[10]. Broadwater and Evans[1] have noted that the Fuoss–Onsager equation, with complete neglect of ion association, leads to a very good fit of the data for unsymmetrical electrolytes to much higher concentrations (greater than 10^{-3} M) than one might expect. Thus, although the theory of this equation, applied to unsymmetrical electrolytes, may be doubtful, it can be treated as an empirical function which will give a good extrapolation to the conductance at zero concentration.

However, for the present work, a least-square treatment of a plot of the molar conductance against $C^{\frac{1}{2}}$ led to the values of $\Delta_m{}^0$ for aluminium ammonium sulphate and potassium aluminiums sulphate which are given in Table 2. Instead of using eqn. (3) to estimate the limiting molar conductance a more precise estimate[11] has been made from the best straight line plot of $1/\Delta_m$ vs $C\,\Delta_m$. The values of the limiting molar conductance at infinite dilution obtained from this method are also given in Table 2.

It is evident from Table 2 that the values obtained by both the methods are in excellent agreement with each other.

In the present case, the Walden product has been calculated by using the $\Delta_m{}^0$ values obtained from

$1/\Delta_m$ *vs* $C\,\Delta_m$. The values of the Walden product are also given in Table 2.

TABLE 2

Limiting molar conductance (Δ_m^o) and the Walden products $\Delta_m^o\,\eta_0$ for aluminium ammonium sulphate and potassium aluminium sulphate in water at different temperatures.

Temperature (K)	Δ_m^o (Onsager)	Δ_m^{o*} ($1/\Delta_m$ *vs*. $C\,\Delta_m$)	$\eta_0 \times 10^3$ (P)	$\Delta_m^o\,\eta_0$
Aluminium ammonium sulphate				
303	280	281	8.007	2.25
308	308	308	7.225	2.23
313	338	337	6.560	2.21
Potassium aluminium sulphate				
303	287	289	8.007	2.32
308	317	318	7.225	2.30
313	345	344	6.560	2.26

$^*\Delta_m^o$ values have been used to calculate the Walden product.

It is evident from Table 2 that the temperature coefficient of the Walden product *i.e.* $d\,\Delta_m{}^o\eta_0/dT$ is negative for both the multicharged electrolytes studied here, which can be attributed to the structure breaking characteristics of aluminium ammonium sulphate and potassium aluminium sulphate in water. In other words it may be said that both the electrolytes behave as structure breaker in water. This conclusion is in excellent agreement with that drawn from molar volume data reported earlier[5]. Further, structure breaking may be attributed to the negative solvation. As the temperature increases, ion-solvent dipole interactions are weakened and there are great chances of decrease in the solvation of ions.

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Study of mechanism of oxidation of ethyl digol by OsO_2 spectrophotometrically

(spectrophotometry/oxidation/osmium tetroxide/digol)

A. K. SINGH, A. PARMAR AND A. K. SISODIA

Chemistry Department, University of Allahabad, Allahabad-211 002

ABSTRACT Kinetics of oxidation of ethyl digol by osmium tetroxide has been studied in alkaline media. A first order dependence with respect to osmium tetroxide concentration has been observed. Sodium hydroxide and ethyl digol exhibit first order kinetics at their lower concentration but both show the retarding effect on the initial rate at their concentrations. A suitable mechanism consistent with the above observations has been proposed.

There have been only very few investigations[1-3] concerning the use of osmium tetroxide as an oxidant. Subbaraman[4] has reported that 3-cyclohexane carboxylic acid reacts with osmium tetroxide in aqueous buffer solution. Anand and Menghani[5,6] have also reported osmium tetroxide as an oxidant in the oxidation of alcohols and diols. In the present study osmium tetroxide has been used as an oxidant in the oxidation of ethyl digol in presence of hydroxyl ion.

Ethyl digol solution was prepared by weighing the appropriate amount of the E. Merck sample of G.R. grade in double distilled water. The standard solution of osmium tetroxide (Johnson Matthey and Co. Ltd.) was prepared by dissolving one gram of its sample in KOH (B.D.H., A.R.). Sodium hydroxide (E. Merck) and KNO_3 (A.R., B.D.H.) grade samples were used. The progress of the reaction was studied by measuring the decrease in absorbance with time at 400 nm (Beckman Spectrophotometer Model-26). This decrease in absorbance corresponds to the decrease in concentration of osmium tetroxide with time.

The first order dependence in osmium tetroxide was established by effecting its manifold variations at the fixed concentrations of other reactants (Table 1). Fig. 1 clearly indicates that the reaction is first order at lower concentrations of both sodium hydroxide and ethyl digol but becomes independent at their higher concentrations. The zero effect of ionic strength (by addition of KNO_3) has been observed. The reaction has been markedly affected by temperature variation.

TABLE 1

Temp. = 26.5°C, [Ethyl digol] = 0.125M, [NaOH] = 0.0370M

$[OsO_4] \times 10^3$M	$-da/dt \times 10^4\ s^{-1}$	Graphical $k_1 \times 10^4\ s^{-1}$	$k_1 = \frac{2.303}{t} \log \frac{a}{a-x} \times 10^4\ s^{-1}$
0.06	2.222	4.826	4.904
0.75	2.777	4.643	4.994
1.00	3.333	5.00	4.746
1 50	5.000	4.807	4.233
2.50	8.230	4.730	4.220
3.30	11.111	4.089	4.239

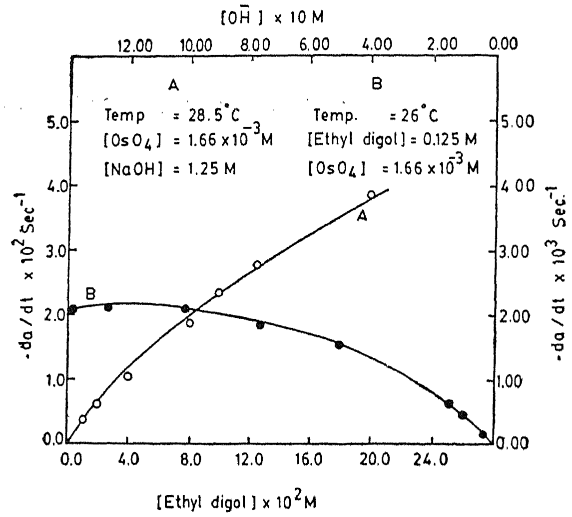

Fig. 1. Variations in the rate constants with the concentrations of ethyl digol (A) and alkali (B).

On the basis of the above results obtained, the following probable scheme can be proposed for the oxidation of ethyl digol :

$$S + OH^- \overset{K}{\rightleftharpoons} S^- + H_2O \qquad \text{(i)}$$

$$\underset{C_1}{[OsO_4(OH)H_2O]^{1-}} + OH^- \overset{K_1}{\rightleftharpoons} \underset{C_2}{[OsO_4(OH)_2]^{2-}} + H_2O \qquad \text{(ii)}$$

$$\underset{C_2}{[OsO_4(OH)_2]^{2-}} + S^- \overset{K}{\rightleftharpoons} \underset{Cx}{[X] \text{ activated complex}} \qquad \text{(iii)}$$

$$[X] \xrightarrow[\text{slow}]{k} \text{Product} + [OsO_3(OH)_3]^{3-} \qquad \text{(iv)}$$

$$[OsO_3(OH)_3]^{3-} + H_2O \xrightarrow{\text{fast}} [OsO_2(OH)_4]^{2-} + [OH^-] \qquad \text{(v)}$$

The reaction of ethyl digol and hydroxyl ion in the step (i) results in the formation of an anion of ethyl digol molecule, which further reacts with the reactive Os(VIII) species yielding the activated complex. The complex thus formed undergoes disproportionation yielding the trihydroxy Os(VI) species and product.

Total osmium concentration which exists in three forms C_1, C_2 and Cx can be written as :

$$[Os(VIII)]_T = [C_1] + [C_2] + [Cx] \qquad (1)$$

The rate in terms of decrease in [Os(VIII)] might be written as

$$-\frac{d[Os(VIII)]}{dt} = 2.k.[Cx] \qquad (2)$$

Considering the equilibrium condition for step (ii)

$$[C_1] = \frac{[C_2]}{K_1\,[OH^-]} \qquad (3)$$

Considering the equilibrium condition for step (iii)

$$[C_2] = \frac{[Cx]}{K_2\,[S^-]} \qquad (4)$$

Substituting the value of C_2 from eqn. (4) in eqn. (3), we get

$$[C_1] = \frac{[Cx]}{K_1 K_2\,[S^-]\,[OH^-]} \qquad (5)$$

On considering eqn. (1, 4 & 5), we have,

$$[Os(VIII)]_T = \frac{[Cx]}{K_1 K_2 [S^-] [OH^-]} + \frac{[Cx]}{K_2 [S^-]} + [Cx]$$

$$\text{or } [Os(VIII)]_T = [Cx]\left\{\frac{1 + K_1 [OH^-] + K_1 K_2 [S^-] [OH^-]}{K_1 K_2 [S^-] [OH^-]}\right\}$$

$$\text{or } [Cx] = \frac{[Os(VIII)]_T K_1 K_2 [S^-] [OH^-]}{1 + K_1 [OH] + K_1 K_2 [S^-] [OH^-]} \qquad (6)$$

On substituting the values of Cx in eqn. (2) from eqn. (6)

$$-\frac{d[Os(VIII)]}{dt} = \frac{2k K_1 K_2 [S] OH] [Os(VIII)]_T}{1 + K_1 [OH^-] + K_1 K_2 [S^-] [OH^-]} \qquad (7)$$

Considering the equilibrium condition for step (i) the equilibrium constant K will be

$$K = \frac{[S^-]}{[S]\,[OH^-]}$$

$$\text{or} \qquad [S^-] = K\,[S^-]\,[OH^-] \qquad (8)$$

On substituting the value of $[S^-]$ from eqn. (8) in eqn. (7), we finally get

$$\frac{-d[\mathrm{Os(VIII)}]}{dt}=\frac{2kKK_1K_2\,[\mathrm{S}]\,[\mathrm{OH^-}]^2\,[\mathrm{Os(VIII)}]_T}{1+K_1\,[\mathrm{OH^-}]\,\{K_2K\,[\mathrm{S}]\,[\mathrm{OH^-}]+1\}}$$

Since $[OsO_4(OH)_2]^{2-}$ is a reactive species of Os(VIII), hence in step (ii) equilibrium is more inclined towards right due to more formation of $[OsO_4(OH)_2]^{2-}$. Under this condition, the following inequality will hold good

$$K_1\,[\mathrm{OH^-}] \gg 1$$

hence eqn. 9 reduces to

$$\frac{-d[\mathrm{Os(VIII)}]}{dt}=\frac{2k\,K\,K_2\,[\mathrm{S}]\,[\mathrm{OH^-}]\,[\mathrm{Os(VIII)}]_T}{1+K\,K_2\,[\mathrm{S}]\,[\mathrm{OH^-}]} \quad (10)$$

This rate law equation clearly explains the first order kinetics with respect to Os(VIII) and first order both with respect to low ethyl digol and hydroxyl ion concentration. The [S] and $[OH^-]$ in the denominator show their retarding effect at higher concentrations.

At lower hydroxyl ion concentration the value of $K\,K_2\,[S]\,[OH^-]$ will be small as compared to 1. Then eqn. (10) reduces to

$$\frac{-d[\mathrm{Os(VIII)}]}{dt}=2k\,KK_2\,[\mathrm{S}]\,[\mathrm{OH^-}]\,[\mathrm{Os(VIII)}]_T \quad (11)$$

At higher concentration of substrate and hydroxyl ion the inequality $K\,K_2\,[S]\,[OH^-] \gg 1$ would be evident and eqn. (11) reduces to

$$-\frac{d[\mathrm{Os(VIII)}]_T}{dt}=2k\,[\mathrm{Os(VIII)}]_T \quad (12)$$

The above equation clearly shows zero order kinetics at higher hydroxyl and ethyl digol concentrations.

A plot of inverse of rate against inverse of hydroxyl ion concentration gives the values of $k\,K\,K_2$ and k as follows

$$k\,K\,K_2 = 0.0470$$

$$k = 0.4545 \times 10^{-2}$$

Similarly the plot between the inverse of the rate and the inverse of ethyl digol concentration gives the values of $k\,K\,K_2$ and k as

$$k\,K K_2 = 0.0307$$

$$k = 0.4545 \times 10^{-2}$$

The k and $k\,K\,K_2$ values obtained by two different methods are quite close and this fair degree of closeness clearly indicates the validity of the rate law eqn. (10) and hence the proposed reaction mechanism.

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Kinetics and mechanism of oxidation of lactose by Nessler's reagent in alkaline medium

(kinetics/lactose/Nessler's reagent/oxidation)

M. P. Singh, R. K. Singh, A. K. Singh, A. K. Sisodia and Madhu Saxena

Department of Chemistry, University of Allahabad, Allahabad

ABSTRACT The kinetics of oxidation of lactose by Nessler's reagent in alkaline medium have been studied. The reaction shows zero order kinetics with respect to initial Hg(II) concentration and first order with respect to reducing sugar. It further follows first order kinetics at low hydroxide ion concentration tending towards zero order kinetics at high hydroxide ion concentration. Retarding trend has been observed throughout the variation of iodide ion concentration. A mechanism has been proposed taking enediol as an intermediate and HgI_3^- as the reacting species.

Goswami *et al.*[1] have studied from an analytical point of view the oxidation of various organic compounds by Nessler's reagent in aqueous alkaline medium. Thereafter, Littler[2] and Halpern[3] studied the oxidation of carbon monoxide and cyclohexanone using mercuric ion as an oxidant in acidic media and postulated a two electron transfer process. The HgO produced was converted to Hg^+ by Hg^{2+} rendering homogeneity to the system. In the present study, we have dealt with the oxidation kinetics of lactose by Nessler's reagent in the presence of sodium hydroxide to propose a mechanism for the reaction and to find out the heterogeneity of HgO formed in the system.

The samples of lactose, mercuric chloride and potassium thiocyanate were of A.R. grade (B.D.H.). Sodium hydroxide and potassium iodide were of E. Merck quality. Solutions of sugar and potassium iodide were prepared fresh before each run. The solution of potassium thiocyanate was standardised according to Volhard's method[4].

The complex (K_2HgI_4) was prepared by mixing the solutions of potassium iodide and mercuric chloride in stoichiometric proportions. The ionic strength was maintained constant by using standard solution of potassium chloride. Aliquots (10 ml each) of the reaction mixture containing the complex (K_2HgI_4), NaOH and KCl were taken separately in conical flasks and immersed in an electrically operated thermostat with an accuracy of $\pm$ 0.1°C. The reaction was initiated by adding the requisite volume of lactose solution to the above aliquots at one minute interval. The progress of the reaction was studied by estimating the amount of Hg(O) produced after definite time intervals. The mercury produced was filtered, washed carefully to remove all other impurities and dissolved in nitric acid. This solution was then boiled to expel the nitrous fumes produced during the formation of mercuric nitrate. The conical flask containing mercuric nitrate solution was then cooled below 10°C and Hg^{2+} was estimated volumetrically by titrating against a standard potassium thiocyanate solution (Volhard's method) using ferric alum as an indicator.

The detailed kinetic study of the oxidation of lactose by Nessler's reagent in aqueous alkaline medium was performed by studying the effect of the variation of different reactants (lactose, Hg(II), OH^- and I^-) involved in the reaction. Table 1 shows that the initial ($-dc/dt$) values for varying Hg(II) concentrations are practically constant, except at very low Hg(II) concentration indicating zero order kinetics with respect to Hg(II). Typical kinetic runs, in the form of straight lines are obtained for each set when the remaining Hg(II) concentration is plotted against time, confirming zero order dependence of the reacinto rate on Hg(II) concentration. The deviation observed in the latter part of the reaction is attributed to the retarding effect of the iodide ion. Low values of ($-dc/dt$) at very low Hg(II) concentration are due to larger availability of free iodide ions as compared to that at higher concentrations.

TABLE 1

Effect of varying Hg(II) concentration on the reaction rate at 30 and 35°C
[Lactose] = 1.0×10^{-2} M, [NaOH] = 4.0×10^{-2} M,
[KI] = 10.0×10^{-2} M, μ = 0.50 M.

Temp. (°C)	$[HgCl_2] \times 10^2$	$(-dc/dt) \times 10^5$ graphical (mole l^{-1} min^{-1})
30	1.25	3.30
	1.00	3.75
	0.67	3.89
	0.50	3.72
	0.33	3.30
35	1.25	7.60
	1.00	8.03
	0.67	8.20
	0.50	7.31
	0.33	7.22
	0.25	7.63

Table 2 shows that ($-dc/dt$) values are increasing in direct proportion with the increase in lactose concentration. Thus, it is apparent that the reaction shows first order kinetics with respect to lactose. The constancy of ($-dc/dt$)/[Lactose] values in column 3 of Table 2, further, confirms first order dependence of the reaction rate on lactose.

TABLE 2

Effect of varying lactose concentration on the reaction rate at 30, 35, and 40°C.
$[HgCl_2]$ = 1.0×10^{-2}M, [KI] = 10.0×10^{-2}M,
[NaOH] = 5.0×10^{-2}M, μ = 0.50M

Temp. (°C)	[Lactose] $\times 10^2$ (M)	$(-dc/dt) \times 10^5$ (mole l^{-1} min^{-1})	$(-dc/dt) \times 10^3$/[Lactose] (min^{-1})
30	0.50	2.00	4.00
	1.00	4.33	4.35
	1.50	7.58	5.05
	2.00	10.67	5.33
	2.50	11.72	4.69
	3.00	13.91	4.64
35	0.50	5.52	11.04
	1.00	10.32	10.32
	2.00	22.40	11.20
	2.50	25.60	10.24
40	0.50	9.66	19.32
	2.00	36.84	18.42
	2.50	55.00	22.00
	3.00	60.00	20.00

The exact dependence of the reaction rate on hydroxide ion concentration (Fig. 1) clearly indicates that the reaction rate follows first order kinetics at low hydroxide ion concentration and becomes almost independent of hydroxide ion concentration at higher concentrations. It is further, observed, that the reaction rate decreases with increasing iodide ion concentrations, indicating retarding effect of iodide ion concentration (Fig. 2).

Before formulating the actual mechanism, it is essential to discuss the chemistry of Nessler's reagent. The reaction between iodide ion and Hg(II) might proceed as follows :

$$Hg^{2+} + 4I^- \rightarrow HgI_4^{2-} \qquad (1)$$

since, the iodide ion concentration has been always kept four times greater than the Hg(II) concentration,

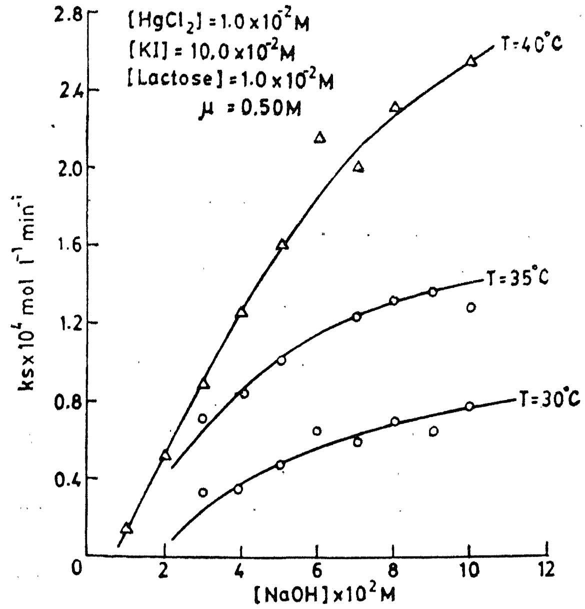

Fig. 1 Plot between reaction velocity and hydroxyl ion concentration.

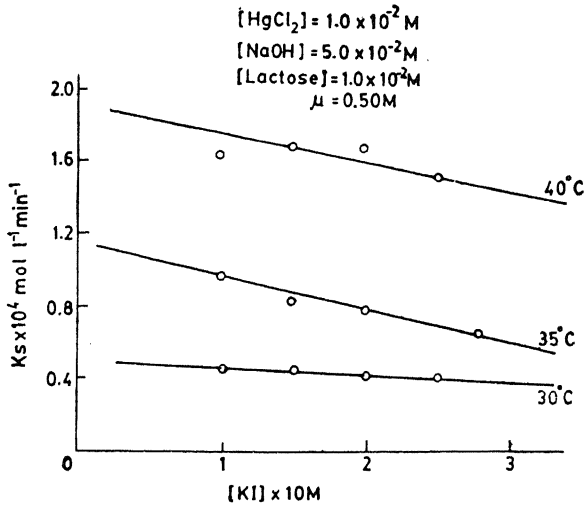

Fig. 2. Plot between reaction velocity and iodide ion concentration.

the main existing species would be HgI_4^{2-}. However, the existence of HgI_3^- has also been reported. According to Moellor[5], in the presence of excess iodide ions, the possibility of the formation of halo complexes other than HgI_3^- and HgI_4^{2-} can be ruled out. Thus, it is quite plausible to assume, that the total Hg(II) in alkaline medium containing excess iodide ions exists as HgI_3^- and HgI_4^{2-}. Kinetically, it appears that there should be an equilibrium of the type shown below in eqn. (2).

$$HgI_3^- + I^- \overset{K}{\rightleftharpoons} HgI_4^{2-} \qquad (2)$$

The retarding effect of the iodide ion shows that the reacting species is HgI_3^- and HgI_4^{2-} is the stable species towards reduction.

From eqn. (2) it is quite evident that the total Hg(II) at any instant would be given by eqn. (3).

$$[Hg(II)]_T = [HgI_3^-] + [HgI_4^{2-}] \qquad (3)$$

Substituting the value of $[HgI_4^{2-}]$ from eqn. (2) in eqn. (3) the value of $[HgI_3^-]$ becomes,

$$[HgI_3^-] = \frac{[Hg(II)]_T}{1+K[I^-]} \qquad (4)$$

In the light of the above facts, a probable scheme of oxidation might be formulated, comprising steps (I-III).

$$S + OH^- \underset{K_{-1}}{\overset{K_1}{\rightleftharpoons}} E^- + H_2O \qquad (I)$$

$$E^- + H_2O \underset{K_{-2}}{\overset{K_2}{\rightleftharpoons}} E + OH^- \qquad (II)$$

$$E + OH^- \underset{\text{fast}}{\overset{K_3}{\longrightarrow}} Hg(O) + \text{other reaction products} \qquad (III)$$

In steps (I-III), S, E^- and E represent lactose, intermediate enediol anion and enediol respectively.

In this scheme of oxidation, it has been assumed that it is the enediol which is being attacked by HgI_3^- in the oxidation step. Considering this to be a plausible scheme of oxidation, the final rate law in terms of the total Hg(II) concentration is given by eqn. (5).

$$-\frac{d\,[Hg(II)]}{dt} = \frac{a\,[S]\,[OH^-]\,[Hg(II)]_T}{b[OH^-]+bk[OH^-]\,[I^-]+C[Hg(II)]_T} \qquad (5)$$

where $a=K_1K_2K_3$, $b=K_{-1}K_{-2}$ and $C=K_3\,(K_{-1}+K_2)$

Eqn. (5) apparently explains the observed kinetics. initially if the inequality, $c\ [\mathrm{Hg(II)}]_T \gg b\ [\mathrm{OH}^-]\ (1 + K[\mathrm{I}^-])$ holds good, eqn. (5) reduces to eqn. (6).

$$-\frac{d\,[\mathrm{Hg(II)}]}{dt} = \frac{K_1 K_2}{(K_{-1}+K_2)}\,[\mathrm{S}]\,[\mathrm{OH}^-] \qquad (6)$$

Eqn. (6) supports the view of zero order kinetics of the reaction rate with respect to Hg(II) concentration in the initial part of the reaction. Further, in a particular run, as the Hg(II) concentration is decreasing with time, simultaneously the factor $b\ [\mathrm{OH}^-]\ (1 + K\ [\mathrm{I}^-])$ is increasing due to gradual liberation of iodide ions. Hence, the above inequality will not be valid and zero order kinetics will therefore, not be observed in the latter part of the reaction.

It has already been established that the reducing sugars give enediol and enediol anions[6,7] in alkaline medium. The catalysis of the hydroxide ion in the oxidation of lactose suggests that the oxidation of lactose takes place in their enediol form. Induction period is due to some time lag in reaching the steady state conditions for the concentration of enediol.

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On simultaneous dual series equations involving Konhauser biorthogonal polynomials

(Konhauser biorthogonal polynomials/dual series equation/Abel integral equations)

A. P. DWIVEDI, B. D. SHUKLA* AND S. C. SHUKLA

Department of Mathematics, H. B. Technological Institute, Kanpur-208 002
**Department of Mathematies, D. B. S. College, Kanpur*

ABSTRACT By using Abel integral equations; we solve simultaneous dual series equations involving Konhauser biorthogonal polynomials.

Dual series equations

$$\sum_{n=0}^{\infty}\sum_{j=1}^{s}\frac{A_{n_j}}{\Gamma(\delta+2\beta+kn_j)}Z_{n_j}^{(\delta+2\beta-1)}(x;k)=f_i(x)\qquad 0\leqslant x<y \quad (1)$$

and

$$\sum_{n=0}^{\infty}\sum_{j=1}^{s}\frac{A_{n_j}}{\Gamma(\delta+\beta+1+kn_j)}Z_{n_j}^{\delta}(x;k)=g_i(x)\qquad y<x<\infty,\ (i=1 \text{ to } s) \quad (2)$$

where $[Z_{n_j}^{\delta}(x;k)]_{n=0}^{\infty}$ is the Konhauser biorthogonal polynomial set, $\beta=0$, $\delta>-1$, $f_i(x)$ and $g_i(x)$ are known functions and A_{n_j} is unkown constant which is to be determined, have been solved.

We require the biorthogonal properties of the Konhauser biorthogonal polynomials[1]

$$\int_0^{\infty}\exp(-x)\,x^{\delta}Z_n^{\delta}(x;k)\,dx=0,\text{ if } m\neq n$$
$$=\frac{\Gamma(1+\delta+kn)}{n!}\quad\text{if } m=n \quad (3)$$

where $\delta>-1$.

The second formula required is the Weyl integral given by Karande and Thakare[2]

$$\int_{\xi}^{\infty}(\exp(-x)(x-\xi)^{\beta-1}Z_n^{\delta+\beta}(x;k)\,dx$$
$$=\Gamma(\beta)\exp(-\xi)Z_n^{\delta}(\xi;k) \quad (4)$$

where $\delta+1>\beta>0$.

The third result that we require is

$$\frac{d}{d\xi}\int_0^{\xi}(\xi-x)^{\beta-1}x^{\delta+\beta}Z_n^{\delta+\beta}(x;k)\,dx$$
$$=\frac{\Gamma(\beta)\Gamma(\delta+\beta+1+kn)}{\Gamma(\delta+2\beta+kn)}Z_n^{\delta+2\beta-1}(\xi;k) \quad (5)$$

where $\delta+2\beta>0$, $\beta>0$, $\delta>-1$.

We have the Riemann - Liouville fractional integral[3] given by Prabhakar[4].

$$\int_0^{\xi}x^{\delta+\beta}(\xi-x)^{\beta-1}Z_n^{\delta+\beta}(x;k)\,dx$$
$$=\frac{\Gamma(\delta+\beta+1+kn)\Gamma(\beta)\,\xi^{\delta+2\beta}Z_n^{\delta+2\beta}(\xi;k)}{\Gamma(1+\delta+2\beta+kn)} \quad (6)$$

where $\beta>0$, $\delta+1>0$.

If $f(\xi)$ and $f'(\xi)$ are continuous in $0\leqslant x<\infty$ and if $0<\beta<1$, then the solutions of Abel integral equations

$$f_1(\xi) = \int_0^{\xi} \frac{F_1(x)}{(\xi-x)^{\beta}}\, dx \tag{7}$$

and

$$f_2(\xi) = \int_{\xi}^{\infty} \frac{F_2(x)}{(x-\xi)^{\beta}}\, dx \tag{8}$$

are respectively given by

$$F_1(x) = \frac{\sin \beta\pi}{\pi} \frac{d}{dx} \int_0^{x} \frac{f_1(\xi)}{(x-\xi)^{1-\beta}}\, d\xi \tag{9}$$

and

$$F_2(x) = -\frac{\sin \beta\pi}{\pi} \frac{d}{dx} \int_x^{\infty} \frac{f_1(\xi)}{(\xi-x)^{\beta-1}}\, d\xi \tag{10}$$

Solution of the Equations:

From (5) and (1), we get

$$\frac{d}{d\xi} \int_0^{\xi} (\xi-x)^{\beta-1} x^{\delta+\beta} Z_{n_j}^{\delta+\beta}(x\,;k)$$

$$\sum_{n=0}^{\infty} \sum_{j=1}^{s} \frac{A_{nj}}{\Gamma(\delta+\beta+1+kn_j)}\, dx$$

$$= \sum_{n=0}^{\infty} \sum_{j=1}^{s} A_{nj} \frac{\Gamma(\beta)}{\Gamma(\delta+2\beta+kn_j)} \xi^{\delta+2\beta-1}$$

$$\times Z_{n_j}^{\delta+2\beta-1}(\xi\,;k)$$

$$= \Gamma(\beta)\, \xi^{\delta+2\beta-1} f_i(\xi).$$

Hence

$$\sum_{n=0}^{\infty} \sum_{j=1}^{s} \frac{A_{nj}}{\Gamma(\delta+\beta+1+kn_j)} \frac{d}{d\xi} \int_0^{\xi} (\xi-x)^{\beta-1}$$

$$x^{\delta+\beta} Z_{n_j}^{\delta+\beta}(x\,;k)\, dx$$

$$= \Gamma(\beta)\, \xi^{\delta+2\beta-1} f_i(\xi) \tag{11}$$

Similarly, from (4) and (2), we get

$$\sum_{n=0}^{\infty} \sum_{j=1}^{s} \frac{A_{nj}}{\Gamma(\delta+\beta+1+kn_j)} \int_{\xi}^{\infty} \exp(-x)(x-\xi)^{\beta-1}$$

$$\times Z_{n_j}^{\delta+\beta}(x\,;k)\, dx = \Gamma(\beta) \exp(-\xi)\, g_i(\xi) \tag{12}$$

Let

$$f_{1i}(x) = x^{\delta+\beta} p_i(x) \tag{13}$$

where

$$p_i(x) = \sum_{n=0}^{\infty} \sum_{j=1}^{s} \frac{A_{nj}}{\Gamma(\delta+\beta+1+kn_j)} Z_{n_j}^{\delta+\beta}(x\,;k) \tag{14}$$

Multiplying both sides of (13) by $(\xi-x)^{\beta-1}$ and integrating with respect to x over $(0,\xi)$ and then differentiating with respect to ξ, we get

$$\frac{d}{d\xi} \int_0^{\xi} (\xi-x)^{\beta-1} f_{1i}(x)\, dx$$

$$= \frac{d}{d\xi} \int_0^{\xi} x^{\delta+\beta} (\xi-x)^{\beta-1} p_i(x)\, dx$$

Now using (9) and (11), we get

$$F_{1i}(\xi) = \frac{1}{\pi} \left(\sin \beta\, \pi\, \Gamma(\beta)\, \xi^{\delta+2\beta-1} f_i(\xi)\right) \tag{15}$$

Again dividing both sides of (15) by $(x-\xi)^{\beta}$, integrating with respect to ξ over $(0, x)$ and then using (7), we get

$$f_{1i}(x) = x^{\delta+\beta} p_i(x)$$

$$= \frac{\sin \beta\pi\, \Gamma(\beta)}{\pi} \int_0^{\infty} \frac{\xi^{\delta+2\beta-1} f_i(\xi)}{(x-\xi)^{\beta}}\, d\xi \tag{16}$$

Let

$$f_{2i}(x) = \exp(-x)\; p_i(x) \tag{17}$$

where $p_i(x)$ is given by (14).

Similarly, multiplying both sides of (17) by $(x-\xi)^{\beta-1}$ and integrating with respect to x over (ξ, ∞) and differentiating with respect to ξ, we get by using eqns. (10) and (12).

$$F_{2i}(\xi) = -\frac{\sin \beta\, \pi\, \Gamma(\beta)}{\pi} \frac{d}{d\xi} \left(\exp(-\xi)\, g_i(\xi)\right) \tag{18}$$

Dividing both sides of (18) by $(\xi - x)^{\beta}$, integrating with respect to ξ over (x, ∞) and then using (8), we get $f_{2i}(x) = \exp(-x)\ p_i(x)$

$$= -\frac{\sin\beta\pi\Gamma(\beta)}{\pi}\int_x^{\infty}\frac{(d/d\xi)\exp(-\xi)\,g_i(\xi)}{(\xi - x)^{\beta}}\,d\xi \qquad (19)$$

from (16) and (19), we write respectively

$$p_i(x) = \frac{x^{-\delta-\beta}\sin(\beta\pi)\Gamma(\beta)}{\pi}\int_0^x\frac{\xi^{\delta+2\beta-1}f_i(\xi)}{(x-\xi)^{\beta}}\,d\xi$$

$$0 < x < y \qquad (20)$$

and

$$p_i(x) = -\frac{\exp(x)\sin(\beta\pi)\Gamma(\beta)}{\pi}\times\int_x^{\infty}\frac{(d/d\xi)\exp(-\xi)\,g_i(\xi)\,d\xi}{(\xi - x)^{\beta}}$$

$$y < x < \infty \qquad (21)$$

The L.H. S. of (20) and (21) are identical, hence multiplying both by $x^{\delta+\beta}\exp(-x)\,Y_{m_j}^{\delta+\beta}(x;k)$, integrating (20) with respect to x over $(0, y)$, integrating (21) with respect to x over (y, ∞); adding and using the orthogonality relation (3), we get, with the help of (14), the solution of the dual series eqn. (1) and (2) in the form

$$\sum_{j=1}^{s} A_{nj} = \frac{1}{\pi}\Big[(n_j)!\sin(\beta\pi)\Gamma(\beta)\int_0^y\exp(-x)\times Y_{n_j}^{\delta+\beta}(x;k)\Big\{\int_0^x\frac{\xi^{\delta+2\beta-1}f_i(\xi)}{(x-\xi)^{\beta}}\,d\xi\Big\}\,dx - (n_j)!\sin(\beta\pi)\Gamma(\beta)\int_y^{\infty}x^{\delta+\beta}\,Y_{n_j}^{\delta+\beta}(x;k)\times\Big\{\int_x^{\infty}\frac{(d/d\xi)\exp(-\xi)\,g_i(\xi)}{(\xi-x)^{\beta}}\,d\xi\Big\}\,dx\Big] \qquad (22)$$

or

$$\sum_{j=1}^{s} A_{n_j} = \frac{1}{\pi}\Big[\sin(\beta\pi)\Gamma(\beta\pi).(n_j)!\Big\{\int_0^y\exp(-x).\times Y_{n_j}^{\delta+\beta}(x;k)\,f_i^*(x)\,dx - \int_y^{\infty}x^{\delta+\beta}\,Y_{n_j}^{\delta+\beta}(x;k).\times g_i^*(x)\,dx\Big\}\Big] \qquad (23)$$

with $\delta + 1 > 0$, $\beta > 0$, where

$$f_i^*(x) = \int_0^x\frac{\xi^{(\delta+2\beta-1)}f_i(\xi)}{(x-\xi)^{\beta}}\,d\xi$$

$$g_i^*(x) = \int_x^{\infty}\frac{(d/d\xi)\exp(-\xi)\,g_i(\xi)}{(\xi-x)^{\beta}}\,d\xi$$

Particular case : If we set $s = 1$ in eqn. (1) and (2) then these reduce to dual series equations involving Konhauser biorthogonal polynomials and our solution (23) is in complete agreement with that of Patil and Thakare[5].

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Age related trend in the skin collagen turnover of *Channa punctatus* Bloch

(aging/collagen turnover/*Channa punctatus*)

MUKHTAR A. KHAN AND A. K. JAFRI

Fisheries Laboratory, Department of Zoology, Aligarh Muslim University, Aligarh-202 001

ABSTRACT Total collagen in the skin of *Channa punctatus* was found to increase with age. Dorsal skin contained higher quantity of total collagen than the ventral. The significance of observed changes has been briefly discussed.

The pattern of growth in poikilothermic vertebrates such as fishes differs from that of mammals in that the former continue to grow almost throughout their life, although the rate of growth gets retarded during the later phase of their life. It is postulated that such a difference in growth pattern is due largely to the turnover of collagen[1], an important protein associated with strength and flexibility of tissue. Collagen changes have been used to assess the actual rate of physiological aging in several vertebrates[2,3] including human being[4].

Studies on fish collagen are relatively few[5]. Gantayat and Patnaik[6] have recorded the age-related trend in the skin and muscle collagen solubility of the murrel, *Channa punctatus*. In the present study an attempt has been made to quantify the total collagen content in the skin of the above species belonging to different age-groups.

Fishes were arranged into length estimates corresponding to age-groups 1^+ to 6^+ as per the method of Khawaja and Jafri[7]. The dorsal and ventral samples were separately analysed for collagen content. The skin portions were stretched and cut into 1 cm^2. These were then hydrolysed in a measured volume of 6N HCl in sealed graduated corning glass tubes at 1.055 kg/cm^2 pressure for 16 h. The content was cooled, neutralized with NaOH and its dilution factor worked out. The hydrolysate was then processed for the estimation of hydroxyproline using the method of Neuman and Logan as modified by Leach[8]. The actual quantity of collagen was calculated by multiplying the total amount of hydroxyproline with the factor 7.46 as per the method of Neuman and Lagan[9].

The total collagen content in both the dorsal and ventral skin samples of *C. punctatus* was lowest in young (1^+ age-group) fish but increased markedly with advancing age. The highest concentration was thus noted in the skin of oldest (6^+ age-group) fish. This increase in the total collagen concentration was slightly less than three-fold in the dorsal and almost six-fold in the ventral skin. The wet weight per unit area of skin, from both the dorsal and ventral portions, was also found to increase with advancing age (Table 1).

Among the unique properties of collagen are its mechanical toughness and chemical stability. These properties are due to the presence of hydroxyproline,

TABLE 1

Collagen content in the skin of *C. punctatus* beloging to different age-gpoups.

Age group Year	Weight of fish (g)	Total length of fish (cm)	REGION					
			DORSAL			VENTRAL		
			Wet wt/ cm^2 of skin (mg)	Total hydroxy proline/cm^2 of skin (mg)	Collagen content (mg/cm^2)	Wet wt. of skin (mg)	Total hydroxy-proline/cm^2 of skin (mg)	Collagen content (mg)
1+	21.51±0.424	12.68±0.059	33±0.577	1.053±0.011	7.85±0.872	12.1±0.458	0.190±0.007	1.41±0.050
2+	51.1 ±0.862	15.81±0.087	55.6±0.760	1.358±0.018	10.13±0.130	15±0.421	0.272±0.006	2.02±0.047
3+	76.5 ±0.969	18.57±0.540	90.5±2.660	1.725±0.021	12.85±0.162	20.6±0.340	0.304±0.005	2.32±0.052
4+	110.05±1.120	20.54±0.540	126.1±1.070	1.817±0.029	13.55±0.218	24±0.258	0.396±0.006	2.95±0.049
5+	127.6 ±2.420	22.61±0.064	136.8±3.274	2.090±0.024	15.59±0.316	27.6±0.718	0.422±0.007	3.19±0.064
6+	269.4 ±4.006	26.8±0.066	181.1±1 410	2 735±0.078	20.01±0 329	32.2±0.840	0 795±0.016	5.93±0.124

Mean ± SE of 10 replicates.

an amino acid occurring in traces in ordinary muscle tissue. Aging seemed characterized by substantial increase in the quantity of this amino acid as is evident from the present observations on the murrel.

The increase in the relative thickness of the skin appeared one of the important manifestations of aging. The increased collagen deposition with advancing age may provide the required mechanical strength and greater resistance to deforming forces.

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Interaction of malathion and mercuric chloride in albino rat

(Interaction/acetylchlolinesterase/Purkinje cells/female rat)

T. S. S. DIKSHITH, S. N. KUMAR, R. B. RAIZADA AND M. K. SRIVASTAVA

Industrial Toxicology Research Centre, Mahatma Gandhi Marg, Lucknow-226 001

ABSTRACT Malathion and mercuric chloride alone and in combination were orally administered to female albino rats to evaluate the acetylcholinesterase activity and the morphological changes. The study has indicated the lack of synergistic effects of the two compounds in animals as revealed by the inhibition of induction of AchE activity and morphological changes in cerebellum.

In India pesticides are used extensively to combat the menace of crop pests and vectors of diseases. A total amount of about 90,000 metric tons of different pesticides has been estimated for the plan period (1984-90) in contrast to consumption of about 500 metric tons during 1950[1]. Among organophosphates malathion is used extensively both in plant protection and in public health programme. Several salts of mercury are used in the treatment of soil and as fungicides and seed dressers. The organic mercurials are also used as seed dressing agents for the prevention of seed borne diseases of vegetables, grains, oilseeds, ornamental plants and fruits[2,3]. Although malathion in view of its high LD_{50} values in rat (oral $LD_{50}=1318$ mg/kg, dermal $LD_{50}=4444$ mg/kg), has been considered as a relatively safe insecticide[3], impurity during manufacture, storage and handling has resulted in human fatalities[4].

Entry of mercury to the animal and human systems through food chain and environmental contamination has stressed the need to know the interaction of mercurial salts with other toxic chemicals like pesticides. This gains further importance in view of the lack of information about the interaction of mercurial salts with other environmental chemicals for extrapolation of epidemiological and toxicological data among population exposed to methyl mercury[5]. Information about the interaction of malathion with other toxic chemicals like mercury is very scanty. The present study therefore, deals with the oral application of malathion and mercuric chloride individually as well as their combination in rats.

One hundred and ninetytwo female albino rats (*R. norvegicus*, Wistar strain) of Industrial Toxicology Research Centre's colony with an average body weight of 200 g were housed in an air conditioned room (75±2°F) of the animal house. The animals were fed with pellet diet (Hindustan Lever Animal Feed, India) and water *ad libitum* throughout the experiment. Treatment schedule of the animals and time interval of killing are shown in Table 1. Malathion suspended in peanut oil and mercuric chloride dissolved in distilled water were orally administered to the test animals. Blood was collected directly from the jugular vein into tubes containing double oxalate solution.

TABLE 1

Treatment schedule of female rats

Treatment/Dose (mg/kg/d)	Number of animals*
Control (Peanut oil) 0 4 ml	48
Malathion (50.0)	24
Malathien (125.0)	24
Mercuric Chloride (0.1)	24
Mercuric Chloride (0.25)	24
Malathion + Mercuric chloride (50 + 0.1)	24
Malathion + Mercuric Chloride (125 + 0.25)	

*Eight animals in each treatment and control groups were killed at 24, 48 and 72 h. after 1, 2 and 3 exposures respectively.

Freshly removed brain was separated into cerebrum, cerebellum and spinal cord and homogenized in 0.25 M ice cold sucrose solution. RBC were separated by centrifuging the blood at 2500$\times$g for 10 minutes. Acetylcholinesterase (AchE 3.1.1.7) was assayed by the method of Hestrin[6].

Paraffin sections of brain were cut at 6 μm thickness and stained in haematoxylin-eosin. Red blood cells (RBC), white blood cells (WBC) counts and haemoglobin contents were determined by the method of Wintrobe and Landsberg[7] and Kolmer *et al.*[8] respectively. Statistical significance of the control and experimental values were calculated according to student 't' test[9].

Rats treated either with peanut oil or mercuric chloride alone or in combination with malathion in both doses exhibited no significant signs of toxicity. However, animals treated with malathion alone at high dose levels showed mild signs of organophosphate toxicity such as tremor, salivation, diarrhoea, dyspnoea but no deaths.

The activity of AchE in different parts of the brain *viz.*, cerebrum, cerebellum, spinal cord and RBC of rats after different treatment is shown in Fig 1.

Malathion (50 mg/kg), mercuric chloride (0.1 mg/kg) alone and their combination (50+0.1 mg/kg) at low dose levels did not suggest any significant inhibition or induction of AchE activity in different parts of the brain in rats after 24 h. However, at 48 and 72 h the chemicals produced varied degree of induction or inhibition of enzyme activity in cerebrum, cerebellum and spinal cord. Malathion (125.0 mg/kg) caused significant inhibition of enzyme activity in different parts of brain after 24, 48 and 72h except in cerebellum (72 h). Mercuric chloride alone in high dose (0.25 mg/kg) also caused varied degree of induction in the enzyme activity (19.71-43.83%). High dose of malathion and mercuric chloride together caused severe inhibition of AchE activity in cerebrum (72 h) and spinal cord (24, 48 and 72h), whereas cerebellum indicated a significant degree of induction of the enzyme activity.

Malathion alone at low dose after 24, 48 and 72 h produced a significant (24.32-65.35%) inhibition of the AchE activity while at a higher dose a much greater inhibition (88.46-95.65%) was observed in RBC of rats. It is of interest to note that mercuric chloride at both dose levels showed an induction rather than inhibition of AchE activity. The degree of induction varied from 13.51 to 81.92 per cent at different intervals in RBC. The activity of AchE after low dose of malathion and mercuric chloride together produced lesser degree of inhibition (16.21-38.31%) whereas the combination of these chemicals at higher dose level caused severe degree of inhibition (73.08-84.38%).

Rats treated with low and high dose levels of malathion, mercuric chloride and their combination for a period of 24, 48 and 72 h caused no gross pathological changes in the vital organs. However, miroscopic examination of the brain of rats exposed to mercuric chloride and malathion together at high

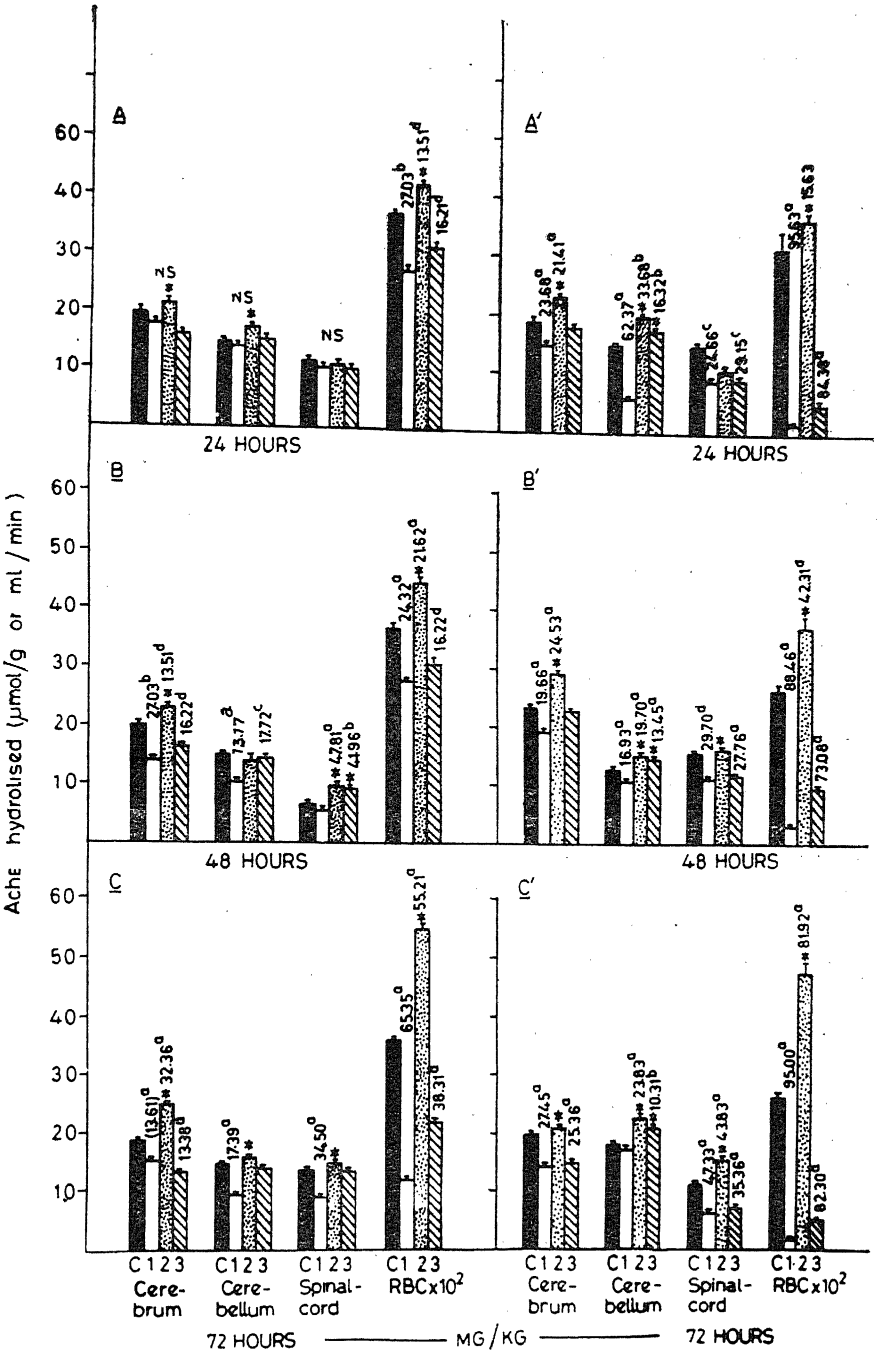

Fig. 1. Histogram showing AchE activity in different parts of brain and REC (μmole/g or ml/min) of rats exposed to malathion and mercuric chloride and their combination; C = control; 1 = Malathion (50 and 125 mg/kg); 2 = Mercuric chloride (0.1 and 0.25 mg/kg); 3 = Malathion + Mercuric chloride (50+0 1 and 125 + 0.25 mg/kg); ABC and A'B'C' represent the lower and higher dose levels respectively; Values on bar diagram indicate the per cent enzyme inhibition; *Represent induction; RBC bar diagram magnified 100 times.

doses showed morphological changes in the cerebellum. The changes were associated with the loss of cells in the granular layer and degeneration of Purkinje cells (Fig. 2). No such cellular changes were found in the cerebellum of rats exposed to malathion alone and in controls (Fig. 3).

There was no significant haematological changes in the treated animals. RBC, WBC counts and haemoglobin contents of the treated animals were comparable with those of normal animals.

The present study has revealed that malathion inhibits AchE activity both in different parts of the brain and RBC. While the lower dose could not elicit significant degree of inhibition of the enzyme, the high dose caused severe inhibition associated with clinical signs of toxicity with slight morphological changes in the brain. Earlier studies with other organophosphorous insecticides like phosphamidon, quinalphos, parathion, methyl demeton and DDVP have also produced similar signs of organophosphorous toxicity and inhibition of AchE activity in RBC and whole brain in species of experimental animals[10-15]. Mercury has caused blockade of membrane transport and disturbed the permeability processes resulting in damage of the vital organs[16,17]. These changes have been traced to the formation of metallothioneine like complex[18,19] Reports have indicated that the binding of this protein is effective particularly after repeated exposure of mercuric chloride[20]. Higher concentrations and chronic exposure of mercuric chloride has produced severe cellular effects and irreversible damage[21].

Mercuric chloride in combination with malathion induced morphological changes in the cerebellum of rats. The changes were associated with the loss of cells in the granular layer and degenerative changes in Purkinje cells. It is of interest to note that similar changes in the cerebellum has been reported with cases of Minamata disease[22]. Affinity of mercurial salts and their accumulation in cerebellum particularly in zones of gray matter is of great concern[23,24]. More studies are needed to know the ultrastructure changes in the Purkinje cells.

Malathion alone could cause inhibition of AchE activity in brain and RBC, its association with mercuric chloride triggered an induction rather than an inhibition of the enzyme. Mercuric chloride in high concentration alone also produced a significant induction of AchE in rats. The induction of AchE activity seems to be due to transient stimulatory effects of mercurial salts. Similar observations have been reported in the *in vitro* studies[25].

Induction of AchE activity in brain after mercuric chloride supports the accumulation of acetylcholine suggesting the competition for binding site(s). There is also a need to know the formation of enzyme inhibitory complexes to elucidate the mechanism of induction of AchE enzyme in animals caused by a variety of xenobiotics. The present study thus indicates, as supported by inhibition or induction of AchE activity and degree of morphological changes in brain, the lack of synergistic effects of the malathion and mercuric chloride in rats. The mechanism of induction of AchE enzyme and formation of enzyme inhibitory complexes due to mercurial salt in animals system needs further study.

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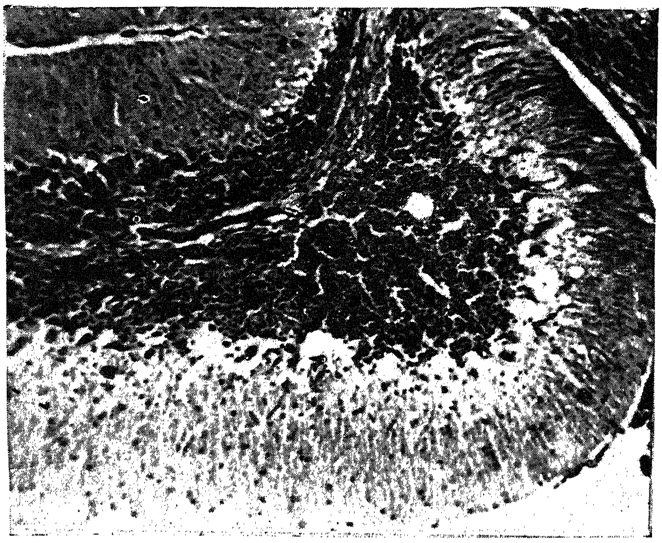

Fig. 2. Section of cerebellum of rats showing loss of cells in the granular layer and degeneration of Purkinje cells. H & E × 133.

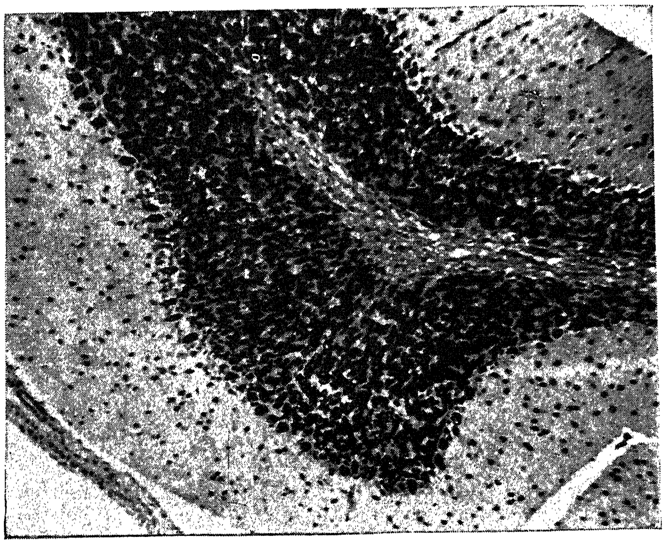

Fig. 3. Section of cerebellum of rats showing normal structure. H & E × 133.

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Fasting-induced implantation failure in mice : Inhibition by α-methyl dopa administration

(nutritional stress/implantation failure/mice/α-methyl dopa/prolactin)

S. C. SAHU AND C. J. DOMINIC

Department of Zoology, Banaras Hindu University, Varanasi–221 005

ABSTRACT Pregnancy failure in newly inseminated mice induced by food deprivation for 48 h at or about the time of implantation was inhibited by administration of α-methyl dopa. Since α-methyl dopa is a potent stimulator of hypophysial prolactin release, the results suggest that the primary endocrine cause of fasting-induced implantation failure is the depression of prolactin release.

The deleterious effects of nutritional stress on reproductive functions in mammals are well documented[1]. Food deprivation during the pre-implantation period causes the death of the blastocysts in mice[2-6]. The hormonal changes during nutritional stress resulting in implantation failure are not clearly elucidated, even though it is suggested that food deprivation may cause a decrease in the secretion of hypophysial gonadotrophin[4] and prolactin[7]. The present study was designed to evaluate the ability of α-methyl dopa, which stimulates prolactin release through suppression of dopamine synthesis, to maintain pregnancy in food deprived mice.

All mice used in the study were outbred albinos belonging to the Parkes (P) strain. They were housed under standard laboratory conditions and maintained on pelleted food (Hindustan Lever Ltd., Ghaziabad) and water *ad libitum*. Females were 10 weeks old virgins. They were paired with stud males; on finding the vaginal plug (day 1 *post coitum*) they were separated from the stud males and housed individually in cages, 40×15×10 cm. Twenty-four hours later (10.00 h on day 2 *post coitum*) the females were divided into three main groups and given the following treatments: *Group I*: Food deprivation for 48 h beginning at 10.00 h on day 2 and injection of α-methyl dopa, 4 mg/female/day (Group IA) or normal saline, 0.1 ml/female/day (Group IB), on days 2 to 6. *Group II*: Food deprivation for 48 h beginning at 10.00 h on day 4 *post coitum* and injection of α-methyl dopa, 4 mg/female/day (Group IIA) and 8 mg/female/day (Group IIB) or normal saline, 0.1 ml/female/day (Group IIC), on days 4 to 8. *Group III*: Left untreated and undisturbed after separation from the stud males.

All injections were given intramuscularly. For administration, α-methyl dopa (Merck, Sharp and Dohme, Bombay) was suspended in normal saline. Females had free access to drinking water during the period of food deprivation. Vaginal smears were examined daily from all females up to day 9 (Group I) and day 11 (Groups II and III) *post coitum*. The incidence of vaginal cornification within these periods was taken as the external manifestation of implantation failure[6]. Females which did not exhibit vaginal cornification were allowed to litter; those which did

not deliver young were presumed to be pseudopregnant.

The results are presented in Table 1. Administration of α-methyl dopa, 4 mg/day, was effective in preventing implantation failure in a large proportion of females in which fasting was started on day 2 *post coitum*. By contrast, the same dose was ineffective in preventing implantation failure in females in which fasting was begun on day 4 *post coitum* (Group IIA). However, when the dose was raised to 8 mg/day, implantation failure was significantly reduced in females (Group IIB). This is consistent with the report[6] that implantation in newly inseminated female mice remains maximally susceptible to nutritional stress on days 4 to 6 *post coitum*. Saline-treated controls exhibited a high rate of implantation failure.

The ability of α-methyl dopa to stimulate pituitary prolactin release is well documented[8,9]. It is suggested that the drug stimulates prolactin release through inhibition of dopamine synthesis by depressing dopa decarboxylase activity[10,11]. The decrease in the rate of nutritional stress-induced implantation failure in mice by α-methyl dopa treatment provides strong circumstantial evidence in favour of the view[7] that depression of hypophysial prolactin release is the primary endocrine factor that contributes to implantation failure in food deprived mice.

The investigations were supported by grants from the Indian Council of Medical Research and the University Grants Commission.

TABLE 1

Effect of α-methyl dopa on fasting-induced implantation failure

Group and treatment	Proportion and (%) of females		
	with implantation failure*	remaining pregnant	remaining pseudo-pregnant
IA. Fasting started on day 2 + α-methyl dopa, 4 mg/day	10/40 (25.0)	25/40 (62 5)	5/40 (12.5)
IB. Fasting started on day 2 + saline, 0.1 ml/day	31/41 (75.6)	10/41 (24.4)	0/41
IIA. Fasting started on day 4 + α-methyl dopa, 4 mg/day	31/32 (96.9)	1/32 (3.1)	0/32
IIB. Fasting started on day 4 + α-methyl dopa, 8 mg/day	15/34 (44.1)	12/34 (35 3)	7/34 (20.6)
IIC. Fasting started on day 4 + saline, 0.1 ml/day	36/36 (100.00)	0/36	0/36
III. Untreated and undisturbed controls	1/23 (4.3)	20/23 (87.0)	2/23 (8.7)

*Significance of differences : IA *vs* IB, $P < 0.001$; IIA *vs* IIC. N S.; IIB *vs* IIC, $P < 0.001$; IA *vs* III, $P < 0.001$; IIB *vs* III, $P < 0.001$.

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ANNOUNCEMENT

Fifth Seminar
on
Solid State Nuclear Track Detectors : Applications to Nuclear Physics

MARCH 24 - 26, 1987

Organised by

Saha Institute of Nuclear Physics
92, Acharya Prafulla Chandra Road, Calcutta-700 009

In collaboration with

Variable Energy Cyclotron Centre
B. A. R. C., CALCUTTA

AND

Department of Physics

Calcutta University

Saha institute of Nuclear Physics (in collaboration with Variable Energy Cyclotron Centre, B.A.R.C., Calcutta and the Physics Department, University of Calcutta) is planning to hold a Seminar on 'Solid State Nuclear Track Detectors : Applications to Nuclear Physics' in March, 1987. This will be the fifth in the series (the earlier ones were organised at BARC, Bombay 1979; PRL, Ahmedabad 1981; Guru Nanak Dev University, Amritsar 1983 and Wadia Institute of Himalayan Geology, Dehra Dun 1985) and will bring together once again the Indian Scientists involved in the use and application of Nuclear Track techniques in various disciplines of basic and applied research. It is hoped that a number of foreign scientists may also participate in the Seminar.

Besides the main theme (Applications in Nuctear Physics), papers are also invited on the following topics :

1. Basic studies : Track formation, mechanism and modelling.
2. Applications to—Cosmic Rays, Space Physics etc.
3. Applications to—Micro Analysis, Radiation Dosimetry, Environmental Sciences, Earth Sciences and Life Sciences.
4. New Detectors and Innovations in SSNTD techniques.
5. Graduate and Undergraduate Laboratory experiments using SSNTDs.

General Information :

1. Abstract volume will be published before the Seminar and the last date of submitting abstracts (two copies) not exceeding 500 words is December 15, 1986.
2. Selected papers after review will be published in the form of Proceedings. Participants are requested to bring completed paper while coming for the Seminar.
3. Guest House/Hostel (dormitory), Hotel Accommodation will be provided to the participants on request.
4. Due to difficulties in arranging accommodation, participants are discouraged to bring families.
5. It wiil be possible to give partial financial assistance especially to deserving research students and participants from the University (provided UGC grants come in time).
6. Next circular will be sent in the month of October, 1986 only to the interested participants who respond to this circular.
7. All communications may kindly be sent to : Professor B. B. Baliga. Organising Secretary, Fifth Seminar on SSNTD, Saha Institute of Nuclear Physics, 92 Acharya Prafulla Chandra Road, Calcutta-700 009.

Application Form

1. Name :
2. Designation :
3. Address :
4. Sponsoring Institute :
5. Tentative Topic of Paper :
6. Hotel/Guest House/Hostel Accommodation (tick the type of accommodation needed) Yes/No
7. Travel Allowance (limited to University Participants) Yes/No
8. Do you support the idea of formation of Nuclear Track Society of India ? Yes/No
9. Suggestions :

Date

Signature

* Kindly give this circular wide publicity.
* Application can be made on plain paper with all the above informations.
* Kindly return the application form to the Organising Secretary.

SERC Winter School on 'Solitons'

(DEPARTMENT OF SCIENCE AND TECHNOLOGY, GOVERNMENT OF INDIA)

The Science and Engineering Research Council (SERC) has formulated a programme of annual Summer/Winter School to encourage research by younger scientists in the frontier areas of Non-linear Phenomena. A Winter School in the series on the topic 'Solitons' will be held at the Bharathidasan University, Tiruchirapalli (T. N.) from Jan 5 – 17, 1987.

It is proposed to cover in the school both the mathematical theory and physical (and biological) applications of solitons. The faculty is likely to consist of the following speakers among others : M. J. Ablowitz, K. Babu Joseph, R. K. Bullough, B. Buti, P. C. Dash, M. R. Gupta, P. K. Kaw, A. Kundu, M. Lakshmanan, R. R. Rajaraman, A. Roy Chowdhury, P. L. Sachdev, A. C. Scott, A. Sen and M. Wadati.

The course is organized by Professor P. K. Kaw (Plasma Physics Programme, Physical Research Laboratory, Ahmedabad) and Professor M. Lakshmanan (Bharathidasan University, Tiruchirapalli).

The Winter School will admit participants on an all India basis. Preference will be given to fresh Ph. Ds. and those working for Ph. D. Degree in soliton theory and its applications and other related areas of non-linear dynamics below the age of 35. Financial support for train travel, board and lodging will be provided to 25 participants; few others who are able to make their own travel and stay arrangement will be admitted.

Completed applications (format appended) should be sent to

Professor M. Lakshmanan

Department of Physics

Bharathidasan University, Tiruchirapalli--620 023

on or before 15 October 1986. Information regarding selection will be sent by October 31, 1986.

Please Post

DEPARTMENT OF PHYSICS

BHARATHIDASAN UNIVERSITY, TIRUCHIRAPALLI-620 023

SERC (DST) WINTER SCHOOL ON 'SOLITONS'

APPLICATION FORM (Model)

1. Name of the applicant :
2. Date of birth, age and sex :
3. Designation and official address :
4. Mailing address :
5. Topics of research and details of research work :
6. (a) Research experience :

   (b) List of publications :
7. Previous participation in seminars/symposia :
8. Academic record (starting from marticulation/higher secondary) :
9. Reference letter(s) (Head of the Department, Supervisor, etc.) :
10. Do you need financial support for

   (a) Travel : Yes No

   (b) Boarding and Lodging : Yes No

Date and Place

Signature of the applicant

Signature of forwarding authority

TATA INSTITUTE OF FUNDAMENTAL RESEARCH

WORKSHOP ON "STUDY OF FAST CHEMICAL PROCESSES"

January 5 to January 9, 1987

January 26 to February 14, 1987

February 16 to February 20, 1987

The Tata Institute of Fundamental Research announces a Workshop on "Study of Fast Chemical Processes". The workshop will be devoted to a review of recent trends in the study of fast chemical processes. Emphasis will be on experimental aspects including design of experiments, analysis of data, modelling, etc. Theoretical methods that are relevant to such studies would also be discussed. Examples for discussion would be chosen from chemical, biochemical, semiconductor and surface sciences.
The workshop will bc divided into three parts :

Session I : January 5 to January 9, 1987

Venue : Tata Institute of Fundamental Research, Bombay

Activity : A laboratory course for 15 young scientists providing hands-on-experience in the following areas :

1. Picosecond spectroscopy
2. Nanosecond spectroscopy
3. T-jump
4. Stop-flow
5. Time Domain Electron Spin Resonance

Session II : January 26 to February 14, 1987

Venue : Lonavla (near Bombay)

Activity : A lecture course covering the following topics :

1. Picosecond chemical dynamics
2. Nanosecond chemical dynamics including pulsed radiolysis
3. Studies with supersonic jets
4. Multiphoton ionization
5. Vibrational relaxation
6. Molecular beams
7. Theory of inelastic scattering
8. Theory of reactive scattering
9. Time domain ESR
10. T-jump and chemical relaxation
11. Stop-flow studies
12. Biochemical applications
13. Ultrafast processes in semiconductors
14. Chemical dynamics at surfaces.

In addition, several seminars on related topics would be arranged. Some of these would be given by participants. The participation in Session II is limited to 50.

Session III ; February 16 to February 20, 1987

Venue : **Tata Institute of Fundamental Research, Bombay**

Activity : **Repeat of Session I for another set of 15 young scientists.**

Faculty. A tentative list of the Faculty for the Workshop is : V. Aquilanti (Perugia); S. Bose (JNU); M. Chowdhury (IACS); S. Doraiswamy (TIFR); P. Felker (UCLA); G. R. Fleming (Chicago); J. W. Gadzuk (NBS, TSA); R. M. Hochstrasser. (Pennsylvania); G. Krishnamoorthy (TIFR); D.H. Levy (MICALO); J. P. Mittal (BARC); P. Natarajan (Madras); J. R. Norris (Chicago); N. Periasamy (TIFR); B. S. Prabhananda (TIFR); R. Ramaswamy (JNU); J. Shah (Bell Labs., Holmdel); J. P. Toennies (MPI-SF, Gottingen); B. Venkataraman (TIFR); A. Weber (NBS, USA)

Sponsorship. Major support for this Workshop has been obtained from

1. Department of Seience & Technology;
2. University Grants Commission;
3. National Bureau of Standards, USA (under the Indo-US (NBS) project on Chemical Dynamics and Laser Spectroscopy);
4. Tata Institute of Fundamental Research

Accommodation. For session II, arrangements are being made to house all participants including the Faculty together. Participants would be required to share rooms. For sessions I and III, accommodation will be provided either in TIFR Hostel or other hostels in Bombay. There is a serious shortage of convenient accommodation in Bombay, and if participants can make their own arrangements in Bombay, it would be appreciated. Applicants for sessions I and III should indicate need for housing, when applying.

Workshop Fees. For Sessions I and III : Rs 500/- per participant for accommodation, food and course material.

For Session II : Rs 5000/- per participant for accommodation, food and course material.

Applicat ons. Applications with particulars of research interests should reach **before November 15, 1986.** Ph.D. Research scholars should arrange for a letter of recommendation. Participants should indicate their desire to attend the laboratory course and express their preference between sessions I and III. For session III, preference would be given to those far from Bombay. It is required that all participants would attend session II in its entirety.

Thanks to support from DST, UGC and NBS, many travel grants and fellowships (for partial support) are available. While applying, intending participants should indicate the support they can receive from their own institutions. Preference for grants and fellowships would be given to young scientists.

ADDRESS FOR CORRESPONDENCE

N. PERIASAMY
CHEMICAL PHYSICS GROUP
TATA INSTITUTE OF FUNDAMENTAL RESEARCH
HOMI BHABHA ROAD, BOMBAY-400 005

Telex : 011-3009 TIFR IN
Cable : ZETESIS: Telephone 219111 (ext. 354)

NOMINATIONS REQUESTED

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Nominations and Inquiries should be sent to :

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Polytechnic University
333 Jay Street
Brooklyn, New York 11201, U.S.A.
(718) 643-5500

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The Marconi Council wishes the Fellowship to be an anticipatory more than a retrospective award. The Fellowship Selection Committee will give special attention to the identification of emerging fields of communications science and technology that are likely to be in the forefront of future applications for the benefit of society.

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Nominations for the Marconi Fellowship should include :

(1) A one-page statement of the reasons for the nomination, considering the criteria above.

(2) The nominee's curriculum vitae, biography and list of publications, not to exceed five pages. *IMPORTANT* : The nominator must supply full biographical information, including a current address for the nominee.

(3) Three letters of support.

(4) Three names and addresses of independent referees who are well acquainted with the work of the nominee.

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(d) All references should be indicated in the text by superscript arabic numerals; e.g. "Mirri[2] while working on.. ". The list of references should be arranged in order of their occurrence in the text. Reference should be given in the following style :

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   Author year Journal vol. beginning page

   White, M. J. D. (1973) *Animal Cytology and Evolution,* 3rd Ed., Cambridge University Press, London, p. 320 (For Books).

   Osgood, C. F. (1977) in *Number Theory and Algebra,* ed. Zassenhaus, H., Academic Press, New York, p. 321. (For edited Books)

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(f) Acknowledgements, if any, should appear at the end of the letter, but just before the references.

(g) Proofs will not ordinarily be sent to authors. If more than 100 reprints are needed, indicate the extra number while sending the manuscript.

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